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Erratum

We have discovered an error in one conclusion of the paper on synGAP by Chen et al. in the May 1998 issue of *Neuron*, "A Synaptic Ras-GTPase Activating Protein [p135 SynGAP] Inhibited by CaM Kinase II" (volume *20*, 895–904). We now find that synGAP activity is not directly inhibited by phosphorylation by CaM kinase II. The experiments depicted in Figure 8 of that paper involved two successive enzymatic reactions. In the first, synGAP in the postsynaptic density was prephosphorylated by CaM kinase II in the presence of Ca^{2+}, calmodulin, and 0.1 mM ATP. Phosphorylated PSD fractions were then assayed for ras GTPase activating activity (GAP activity). Pyrophosphate, a potent inhibitor of phosphatases in the PSD fraction, was added to the GAP assay to preserve the phosphorylation state of synGAP. Because of a fault in the design of the experiments, controls were not included in which *both* ATP and pyrophosphate were present in GAP assays containing nonphosphorylated synGAP. We have found that the combination of residual ATP (30 μM) from the prephosphorylation reaction and 6 mM sodium pyrophosphate is sufficient to inhibit the GAP activity of synGAP (panel A). No additional inhibition is produced by phosphorylation of synGAP by CaM kinase II (panel B). The error was reinforced by an experiment shown in Figure 8A of the paper that indicated inhibition of synGAP activity was blocked when inhibiting antibodies against CaM kinase II were included during the prephosphorylation reaction. We cannot reproduce blockade of synGAP inhibition by these antibodies. In light of this correction to Figure 8, the paper would be more appropriately titled "A Synaptic Ras-GTPase Activating Protein Phosphorylated by CaM Kinase II." We apologize for any confusion caused by this error.

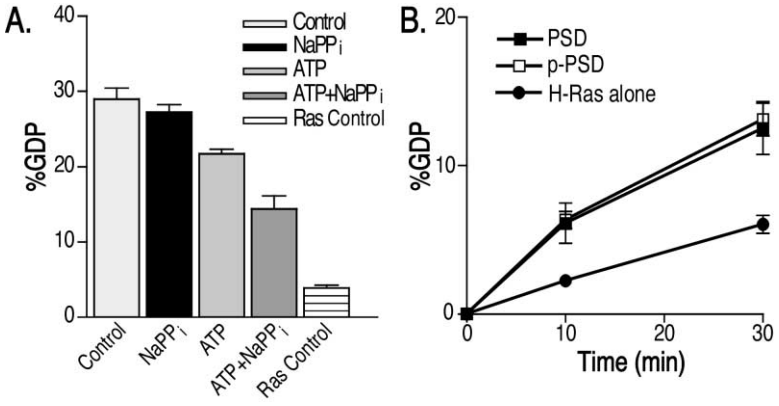

Figure 8. (A) SynGAP activity is strongly inhibited by the combination of ATP and pyrophosphate. GAP assays were performed for 30 min in the presence of 30 μg of PSD protein. ATP (30 μM), Na pyrophosphate (6 mM), or both were added as indicated. Ras Control contained no PSD protein. Assays were performed as described in Chen et al. (1998), except that 10 mM EDTA was added at the end of the assay to reduce nonspecific hydrolysis of GTP.

(B) Phosphorylation of synGAP by CaMKII produces no additional inhibition of synGAP. PSD protein (30 μg) was incubated with 0.1 mM ATP in the absence (PSD) or presence (p-PSD) of 0.3 mM Ca^{2+}/0.6 μM calmodulin as described in Chen et al. (1998). Reactions were stopped by addition of EGTA and NaPPi. GAP assays were initiated by addition of [^{32}P]GTP-bound Ras as described in Chen et al. (1998). The final concentration of ATP was 30 μM and of NaPPi was 6 mM.

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